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Burn Care: The Use of CPM, Electrical Stimulation and Lasers

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University of North Dakota

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BURN CARE: THE USE OF CPM, ELECTRICAL STIMULATION
AND LASERS

by

Jennifer Walter
Bachelor of Science in Physical Therapy
University of North Dakota, 1993



An Independent Study

Submitted to the Graduate Faculty of the

Department of Physical Therapy

School of Medicine

University of North Dakota

in partial fulfillment of the requirements

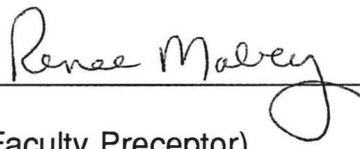
for the degree of

Master of Physical Therapy


Grand Forks, North Dakota

May
1994


This Independent Study, submitted by Jennifer Walter in partial fulfillment of the requirements for the Degree of Master of Physical Therapy from the University of North Dakota, has been read by the Faculty Preceptor, Advisor, and Chairperson of Physical Therapy under whom the work has been done and is hereby approved.



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ABSTRACT

The treatment of patients with burns has always been a tedious venture. The burn team may devote many long and painful hours to the patient's rehabilitation. Physical therapists play an important part on this team, especially in the areas of wound care and pain control. The purpose of this literature review is to analyze the physiological mechanisms of continuous passive motion, electrical stimulation, and lasers in regard to wound healing and pain perceptions.

In relation to wound healing, electrical stimulation and lasers were found to enhance the actual healing process, mostly in the early phases of healing. Continuous passive motion (CPM) devices have been shown to assist in the re-alignment of collagen fibers, thus making the wound stronger.

All three modalities (CPM, electrical stimulation, and lasers) are thought to control pain by the same mechanism, the Gate Control Theory. In addition, electrical stimulation may manage pain by causing the release of endogenous opiate, endorphins, and enkephalins.

In conclusion, these modalities have been shown to have some positive effects on the areas of wound healing and pain control. These findings have been inconsistent in the recording of parameters and overall outcomes when

compared to controls. It is recommended by the author that more research in these areas be completed in the U.S. and that consistency of recording become standard.

CHAPTER I

INTRODUCTION

It is estimated that each year over two million people in the United States are burned.^{1,2} Burn injuries are second to motor vehicle accidents as a cause of accidental death among Americans.¹ These deaths are most often related to infection of the wound.³ Of these victims, 41% are under 19 years of age. Two-thirds of all burns occur in the home; the elderly are of the highest percentage of people burned at home.¹

This chapter will introduce physical therapy's role in the care of a patient who has sustained a burn. It will explain the history of physical therapy intervention, the phases of burn care, and the physical therapist's role in those phases. Finally, this chapter will state the purpose for the chapters that follow.

History of Physical Therapy Involvement with Burns

According to Richard, Staley, and Finley,⁴ therapeutic medical intervention with this patient population dates back at least three centuries. In 1607, Fabricius Hildanus treated burns by splinting to prevent contracture. Turner, in 1790, stated, "one must use all one's art to maintain the most favorable position and the most useful motion." Europe opened the first burn unit under the direction of James Syme in 1848. Early physical therapy for the

burn patient was advocated by Moncrief in 1958. Gronley restarted ROM six to eight days post graft in 1964. Larson came out with the first customized JOBST pressure garment in 1972.⁴

Treatment of patients with burns has evolved greatly since its beginnings. Physical Therapy, once a secondary intervention, is now an integral part of the burn team from the acute phase of care.

Phases of Care

There are three phases of burn care: emergent care, acute care, and rehabilitation.⁵ Emergent care is the first phase. It begins at the site of the accident and consists of life-saving techniques to help the critically injured.

Acute care is stage two. It starts from the admission to the hospital and continues until the burn wound is closed. An aggressive physical therapy program begins in this phase to prevent as much loss of function as possible.

Rehabilitation is the final phase. It picks up at the end of the acute phase, when the wound is closed, and continues to the point when the patient has reentered and readjusted to his/her community. This phase may last up to two years.

Roles of Physical Therapy

Physical therapy should start within 24 to 48 hours post burn.¹ The physical therapist's role is to identify immediate or potential problem areas and formulate treatments to counteract them. Evaluation and reevaluation of wound

size, pain, and scar management, ROM, strength and endurance, ADLs, and functional activities are vital areas of care for burn patients.^{6,7,8}

To accomplish all of the treatment goals set by the evaluation, specific techniques and modalities are employed by physical therapists. Some of the more common techniques include ROM, exercise, positioning, splinting, and debridement.

There are other physical therapy techniques not commonly used with the burn population that may be of benefit for these patients. Continuous passive motion (CPM), electrical stimulation, and lasers are modalities that could be used to enhance healing and reduce some of the great discomfort that is experienced in the burn situation.

Statement of Purpose

There are four main purposes for this paper. These are:

1. To describe the mechanism of wound healing
2. To describe the mechanism of pain
3. To describe physiological responses to CPM, electrical stimulation and lasers
4. To describe how those responses can benefit the patient in terms of wound healing and pain management.

CHAPTER II

WOUND HEALING

To see how the CPM, electrical stimulation, and lasers can be of benefit, it is imperative to understand the basics of wound healing. This chapter will explain the types and characteristics of burns, the phases of wound healing, and factors that are unique to burn wounds.

Types of Burns

Burns are categorized by their primary mechanism of injury.^{8,9} The most common burn is the thermal burn, caused by flames, semiliquids/semisolids, liquids, and solids. Burns occur when the cellular temperature rises so high that the heat absorption is greater than heat dissipation and survival of the cell is not possible. The time of exposure required to cause a burn varies with temperature. For each degree rise in temperature between 44 degrees and 51 degrees Celsius, the rate of cellular destruction doubles. Only a brief exposure to higher temperatures produces tissue destruction. At 71 degrees Celsius, a one second exposure produces a full-thickness injury.^{9,10}

The second type of burn is the electrical burn. The body acts as the conducting medium through which the electric current travels. The amount of damage caused by the electrical current depends on six factors: 1) type of

current, 2) voltage, 3) amperage, 4) duration of contact, 5) resistance of tissues, and 6) the pathway of the current through the body. Different body tissues exhibit different amounts of resistance to current. The tissues with the greatest to least resistance are: bone, fat, tendon, skin, muscle, blood vessels, and nerves.⁷ The tissues that exhibit the least resistance are most likely to have greater tissue damage. There can be extensive unseen internal damage and an entrance and exit wound.

The final type of burn is the chemical burn, caused by strong acids and alkalies. Chemicals cause tissue destruction by denaturing the tissue protein or interfering with cell metabolism.⁹ Water is the best neutralizer and wounds are thoroughly washed. However, the chemical continues to penetrate the tissues; only after 72 hours can the true tissue damage be assessed.⁷

Extent and Depth

The extent of body surface the wound covers can be determined in various ways. The "Rule of Nines" chart^{1,6,7,9,11} (See A) classifies the body into percentages of nine or eighteen percent. This is a very quick and easy chart to use, but it does not adjust for children except infants.

The Lund-Browder chart^{1,6,7,9} (See A) measures the total body surface area (TBSA) burned. It breaks body parts down into percentages that differ with age. Both methods are useful objective tools for continuous reevaluation of the wound area.

The depth of the wound can be described as superficial, partial-thickness, or full-thickness.^{6,7} These depths are differentially diagnosed by the wound color and vascularity, surface appearance and pain, and swelling, healing and scarring.

Phases of Wound Healing

Once an insult has occurred to the skin, there are three phases to the healing of the wound. These are inflammation, proliferation, and maturation.

Inflammatory Phase

The first phase is called the inflammatory phase, also known as the lag phase.¹² Sources vary on the length of this phase, but it starts at the time of injury and lasts for three to five days.^{6,13} The main purpose of the lag phase is to prepare the wound for repair. Its major functions are to rid the wound of foreign tissue and fight infection.^{12,13} Three reactions (vascular, hemostatic, and cellular) happen in this phase.

The vascular reaction, which has the task of stopping the hemorrhage, contains initially a vasoconstriction. This immediate constriction of the blood vessels in the area is caused by the release of local mediators, such as norepinephrine, and happens in the first five to ten minutes after the insult. A more prolonged vasoconstriction is continued by the mast cells and serotonin that is released when the platelets are released into the interstitial fluid. This constriction is followed by a vasodilatation when the mast cells, basophils and platelets release histamine. Thus begins the edema formation in the wound.

The hemostatic reaction begins when the exposed collagen of the wound triggers the release of platelets into the wound area. They aggregate and form a platelet plug to stop the bleeding.

The cellular reaction is the final reaction of the inflammatory phase reactions. Leukocytes are necessary for the cleaning out of debris in this phase. Within one hour after the onset of inflammation neutrophilic margination occurs, whereby the entire endothelial margin of venules is covered with neutrophilic leukocytes. Monocytes cells also travel through the vascular system to arrive at the wound. The monocytes are vital to healing because they are responsible for the phagocytosis that removes the foreign material from the wound area. Mast cells release heparin and histamine, as already mentioned above, and contribute to the vasodilation of the wound area. Other active substances include the kinins which increase the microcirculation and prostaglandins that also increase the permeability of the vascular system.¹³ When thinking of the inflammatory response, it is important to remember this is a complex process of interrelated events.

Proliferative Phase

The proliferative phase begins about day three post injury and lasts up to day 20. Three events occur in this phase: collagen formation, reepithelialization, and wound contraction. These events are all tied together and will be discussed as such. For the reestablishment of tissue in the wound, fibroblasts migrate into the wound area. This is called fibroplasia. The

fibroblasts form a connective tissue matrix.¹⁴ After the connective tissue matrix is formed, collagen synthesis begins, which is needed for the normal repair of the wound site.

Reepithelialization begins within hours of the insult. What causes it to begin is unclear. The "free edge effect," or loss of cellular contact, is thought to stimulate the migration of epithelial cells across the wound surface.^{6,14}

As the proliferative phase begins to wind down, wound contraction peaks. The decrease in size of the wound is due to the fibroblastic activity. Contraction of the wound continues into the third and final phase of wound healing.

Maturation

Maturation,⁶ or remodeling^{14,15} as it has been called, is the final phase. It begins about day nine post injury and can last up to two years.^{14,15} Now is when the fibroblasts disappear. The collagen that was haphazardly formed in the proliferative phase is now taking on some alignment. Wolff's law, which will be discussed in detail later, becomes a factor. This law briefly states that a tissue that is strained will increase in strength.¹⁶ A word of caution: the scar will only regain 80 percent of the original tensile strength of normal skin.⁶

These phases proceed as described above with a healthy patient and a healthy wound healing environment. It is also important to remember it is difficult to separate the phases. They overlap in sequence.

Factors Unique to Burn Wound Healing

Burns are unique wounds in many ways. The burn wound is difficult to classify because it is seldom uniform. The wound has three zones: coagulation, stasis, and hyperemia.^{6,17} The zone of coagulation is the most badly injured area of the burn with irreversible destruction. There is no blood supply and the tissue is dead.

The zone of stasis is the "in-between" area of the burn. This is the most critical tissue of the wound; it can be saved or easily lost. With an ideal healing environment, the tissues recover. In a less than ideal situation, the partial-thickness defect may quickly become a full-thickness problem.

The most outer area of the burn wound is the zone of hyperemia. This area outlines the edge of the wound and is next to normal tissue. It is reddened with erythema.

Burns are uniquely dynamic. Often, a burn has a large amount of dead tissue that remains in place. This dead tissue may be sterile but can easily change to an excellent bacterial culture medium. After a patient becomes medically stable, the wound itself presents the greatest threat to his or her survival.⁹

Burn wounds are different from other traumatic wounds in two other ways. A burn leaks copious amounts of water, serum, and blood, which can cause many secondary health problems. Secondly, the wound remains open for a long time, lending itself to an increased chance of infection.

This chapter explained the events that occur in the three phases of wound healing. It also explained how burns are unique wounds and how they are classified by type and their zones. The next chapter will discuss how pain is perceived from the burn wound that is healing.

CHAPTER III

PAIN PATHWAYS AND MODULATION

Wound healing is not the only event unique to the burn population. Pain caused by an uncovering of free nerve endings is also special to these patients. As physical therapists, most of our patients approach us with a complaint of pain. In the patient with a burn, it is a feeling that may take a long time, if ever, to subside. This chapter will focus on the internal pathways pain takes from stimulus to perception and on the modulation of those pain pathways to help control pain.

Nociceptors

Nociceptors are the primary cutaneous afferents that receive and transmit information when a noxious stimulus or tissue damage is occurring.¹⁸ These receptors are not uniformly sensitive and they are put into several categories depending on their responses to mechanical, chemical, and thermal stimulation and their conduction velocities. In patients with burns, there are two main receptors that signal pain, A-delta and C polymodal nociceptors.

A-delta nociceptors are mostly distributed superficially in the skin. These fibers are small in diameter and myelinated. They carry the message of a

noxious stimulus at about 15 meters per second (m/sec).^{18,19} Their transmitter substance is unknown.

A-delta afferents are divided into heat nociceptors and high-threshold mechanoreceptors by Mayer and Price.¹⁹ These authors feel that A-delta heat nociceptors are the only myelinated primary afferents innervating the skin and extremities which can be reliably activated by noxious heat. The high-threshold mechanoreceptors will respond only to stimulus intensities that produce overt tissue damage, which is not typical of noxious heat. Although Mayer and Price differentiate between these two A-delta fibers, both fibers respond to noxious or potentially damaging skin stimuli with the highest impulse frequency.^{20,21}

C polymodal nociceptors are unmyelinated fibers found in the deeper layers of the skin and in almost every other tissue except for the nervous system itself. These are better known as "free nerve endings." They are sensitive to factors common to tissue damage, such as noxious heat, mechanical or chemical stimuli. To convey their impulse, these fibers contain a characteristic peptide called Substance P. The message sent by C polymodal fibers travels at one m/sec, slower than the A-delta fibers.^{18,19}

A-delta and C polymodal fibers have been related to two different sensations of pain, first pain and second pain. First pain is the initial sharp pain that is very well localized. This sensation does not last as long as the noxious stimulus that caused it. The A-delta fibers are thought to be involved with this first pain.^{18,19}

A few seconds after the first pain, the second pain begins. It is a burning, throbbing sensation that is less localized. The C polymodal fibers are stimulated in this situation. This stimulation lasts longer than the initial stimulus and summates with repeated stimulation or tissue damage.^{19,22} This phenomenon may partially explain the hypersensitivity of injured tissue.

Pain after a burn injury could be explained best as second pain. In partial thickness burns, free nerve endings are exposed and constantly being stimulated. But a patient with third degree or full-thickness burns will not feel pain because the nociceptors have been destroyed.

The movement of the impulse up the axon of the A-delta fibers is to their cell body located in the dorsal root ganglia. Seventy percent of the C polymodal fibers also have their cell bodies located there. The other 30% of C fibers double back to the mixed nerves and enter the spinal cord or the brainstem through the ventral (motor) root.

Central Pain Mechanisms and the Brain

The dorsal horn of the spinal cord is the more posterior layers of grey matter. Grey matter is divided into nine laminae or layers.^{22,23} (There are ten layers if the grey matter surrounding the central canal is included.)

There are two principles governing grey matter and its layout in the spinal cord.²² First, each lamina contains several morphologically and functionally different types of cells whose characteristics are not homogeneous. Second, cells of each lamina only send axon branches deeper in the grey matter.

The dorsal horn is made up of Lamina I - VI and receives primary afferents from the periphery. Lamina I (L.I) is the marginal zone, with cells bordering the tip of the dorsal horn. Lamina II is deep to L.I and is called the substantia gelatinosa (SG). (The lack of myelination in L.II gives it a gelatinous appearance, hence its name.) Lamina III - VI are called the nucleus proprius.²² Lamina VII and VIII lie deep to the dorsal horn and are called the intermediate grey matter. Interneurons in Lamina VII and VIII do not receive or project information outside of the central nervous system; they input and output to the superficial lamina or axons descending from higher brain centers. Finally, Lamina IX contains motor neurons and axons leaving the spinal cord, that innervate muscles in the periphery. This region is called the ventral (anterior) horn.²²

Small myelinated fibers (A-delta) along with a minority of unmyelinated fibers (C polymodal) terminate in L.I and in part of L.V. It is important to note, L.I cells receive most of their input from nociceptors and respond only to nociceptive input. Thus they are said to be "nocispecific."²² The remainder of the C polymodal fibers end in L.II.^{22,23}

Cells in L.V show convergence of input from both nociceptive and non-nociceptive (innocuous) primary afferents. These have been named wide dynamic range (WDR) cells. They respond to both nociceptive and innocuous stimuli, but with a different pattern of impulses.^{19,22}

The lateral spinothalamic tract (LSTT) is the major pathway for the relay of noxious input from the spinal cord to the brain. Once the nociceptors are activated by tissue damage, they send their impulses mainly through the reticular formation and the intralaminar thalamus to the whole cerebral cortex. Most of this information is directed to the prefrontal regions, where it is perceived as pain.²²

Modulation of Pain

Once the pathways of pain are understood, its modulation can be explained in easy terms. Three types of modulation will be touched upon, peripheral modulation, opiate substances, and the Gate Control Theory.^{19,23,24}

The only known example of peripheral modulation is the effect of taking aspirin. This causes an increase in the threshold of peripheral nociceptors. All other forms of modulation take place in the central nervous system.²⁴

Within the central nervous system, endorphins and enkephalins play a primary role. These substances act in different manners and their origin is largely unknown. Enkephalins act like a neurotransmitter to modulate pain at the spinal cord level, causing inhibition of pain in the substantia gelatinosa.²⁴ They are quick in response to noxious stimuli and short in effect. Endorphins, on the other hand, are more like a hormone. They are thought to be released into the blood and thus take longer to act; their effects are for a greater duration than enkephalins effects.^{19,23} Once in the bloodstream, endorphins circulate throughout the central nervous system modulating pain.

The third type of pain modulation is the Gate Control Theory. This theory was introduced by Melzack and Wall in 1965.^{19,20,22} It postulates that within the spinal cord there are mechanisms which may "open the gate" to impulses generated by noxious stimulation, so we become aware of them, and others which tend to "close the gate" so we are less aware of noxious input. A prime example is "rubbing" a pain better. In more scientific terms, it appears that low-threshold cutaneous mechanoreceptors (A-beta fibers) are at work. Their main central axons, which pass up the dorsal columns without synapse until they reach the gracile and cuneate nuclei, give off collaterals upon entering the spinal cord. These collaterals terminate on the A-delta and C polymodal nociceptor fibers in the outer laminae of the dorsal horn. Activation of A-beta fibers, by rubbing the skin or applying electrical stimulation, partially stimulates the nociceptor terminals. When the noxious impulse comes along, the terminals are in a refractory state and their response is decreased or abolished. This activity, also called presynaptic inhibition, decreases the stimulation and consequent perception of pain in the cortex.^{24,25}

The theories for pain perception and modulation described in this chapter are still not fully understood. Much like other processes in the body, they are still being researched. This chapter presented the present view of the processes of pain. The Gate Control Theory is the predominant mechanism whereby continuous passive motion, electrical stimulation, and lasers assist in

pain control. The exact rational behind each modality will be discussed in each respective chapter.

CHAPTER IV

CONTINUOUS PASSIVE MOTION AND BURNS

The natural healing process is a continuous, 24-hour event. With this in mind, it makes sense that the passive range of motion done in a 30-minute session of physical therapy is not enough to prevent tissue adherence and loss of motion. Nor does 30 minutes of range of motion assist with the alignment of the newly formed collagen fibers.

This chapter will explain four key areas: the history and design of continuous passive motion (CPM) devices, the rationale behind the use of CPM devices with the burn population, Wolff's law in relation to CPM devices and wound healing, and how CPM devices have been shown to decrease pain.

History and Design

Dr. Robert Salter is accredited with introducing the concept of continuous passive motion in 1970.^{26,27,28} But the first documented use of a guided motion system was at Rancho Los Amigos in 1960.²⁷ Its application was to passively flex and extend the knees of post-synovectomy rheumatoid arthritic patients.²⁷

Continuous passive motion devices have been shown to maintain or increase ROM, decrease joint stiffness, decrease pain, accent healing, and enhance nutrition to joint areas.²⁹ With the labor-intensive nature of burns, CPM

devices could be of great use. At the current time, there are CPM devices manufactured for all major joints of the body.^{26,30} There are two basic approaches used to design a CPM device, anatomical or free-linkage. Each has its own philosophy. The anatomical design supports and mobilizes the joint as similar to natural anatomic motion as possible. The free-linkage design allows the joint to seek its own anatomical motion.^{26,31} The anatomical design gives greater patient comfort,²⁶ thus would be a better choice for patients with burns.

Rationale for CPM Usage

Covey et al³² completed a study on the efficacy of using CPM with hand burns. The authors were able to identify certain patient types that would or would not benefit from using CPM devices. There are three groups felt to benefit from CPM.^{30,32}

The first group includes individuals with extensive involvement to multiple kinetic areas. For instance, a patient with 60 percent TBSA burned would most likely have all major joints involved. Using a CPM would allow maintenance of ROM to all areas and let the therapist give more attention to other critical areas.

A second group to benefit from CPM usage would be patients who are unable to actively participate in their rehabilitation. This group includes patients with decreased cognitive states. It also includes individuals whose burn has compromised the metabolic system and whose fatigue is a factor in completing therapy sessions. Richard et al^{33,34} studied the physiological responses of a

patient with a 39 percent TBSA burn; "done safely" CPM did not induce any hazardous physiological stress.

The third group thought to benefit from CPM are those patients who have difficulty participating in therapy because of pain or anxiety. The CPM can be adjusted to any amount of motion within the normal limits of the joint. This allows the anxious or painful patient to increase the setting for ROM when they are comfortable.

There are, however, two problems with CPMs that limit their capabilities.³² A one-size-fits-all design makes using the CPM with grossly edematous burns almost impossible. A CPM is also not appropriate for the combative patient as injury to self or others may occur. The positive and negative aspects must be weighed for each patient before deciding whether or not to use a CPM device.

Wolff's law

Wolff¹⁶ notes that the structure of bone is related to the function it performs. A bone's internal structure changes to fit the changes in functional demands.¹⁶ Researchers^{16,26,29,30} have applied Wolff's law to other tissues of the body; the principle applies also to soft tissue healing. The tensile strength of a healing wound is initially related to the collagen content within in the wound. After two to three weeks, further gains in tissue tensile strength are made by chemical and physical changes within the collagen.¹⁶ Physical tension on the collagen fibers by wound contraction, muscle contraction, or external

forces, such as CPM devices, realigns the fibers to better accommodate the stress required for that anatomical area.¹⁶

Wolff's Law, as it applies to soft tissues healing, gives support for the use of CPM with the burn population. CPM also applies tension to the wound and is very beneficial in strengthening the wound.^{27,30,35} Used early, CPM devices can assist in aligning collagen fibers, thereby helping to maintain the proper motion of an area.

CPM and Pain

Continuous passive motion helps decrease pain. While the exact mechanism for decreased pain is unknown, the most probable explanation is Melzack and Wall's Gate Control Theory.^{19,20,22,30} The CPM's mechanical stimulus provides constant afferent pulses sent from the A β fibers to the brain. The impulses "close the gate" and block the transmission of pain from the C fibers.^{19,20,22,30}

Continuous passive motion used with the burn population can only compliment other treatments performed by the physical therapist. Also it will assist in making the wound stronger. It will make the other treatments, like dressing changes and debridement, more tolerable due to the overall decrease in pain. The following chapters will discuss how electrical stimulation and lasers have been found to affect the areas of wound healing and pain.

CHAPTER V

ELECTRICAL STIMULATION

Electrical stimulation is the second modality frequently used by physical therapists to enhance wound healing and control pain. Its use in these areas has been well documented. Various forms of electrical stimulation have been cited as assisting in wound healing via infection control, cellular proliferation, fibroblastic formation, wound contraction, collagen synthesis, improved circulation, decreased hypoxia, decreased edema formation, and pain control.^{36,37,38} This chapter will focus on electrical stimulation with respect to the forms of stimulation and their effects.

Forms of Stimulation and Their Effects

The earliest documentation of electrical stimulation for wound sites was published by Sir Kenelm Digby in 1688.^{39,40} Smallpox lesions were healed without scarring after application of a charged gold leaf. Interest in this method of treatment was sparked again in the 1960s, when Kanof³⁹ used the gold leaf technique for wounds resistant to healing. This proved effective for accelerating the healing of pressure sores but "produced excessive granulation tissue formation when used in treating burn wounds."^{39,40} Thus most of the studies

related to electrical stimulation of wounds deal with ulcers, pressure sores, and other lesions that are more resistant to healing.^{41,42}

Low Voltage, Constant Microamperage, Direct Current

Low intensity direct current (LIDC) was one of the first off-shoots of the gold leaf findings. LIDC is deliverance of less than 50 volts through a negative electrode called a cathode or a positive electrode called an anode.³⁹

In 1968, Assimacopoulos⁴³ used the ears of four rabbits to test his theory of assisted wound healing. He created full-thickness defects. Wounds of two rabbits were exposed to negative current continuously until the healing process was completed. This process took only 18 to 19 days, almost seven days faster than the two control rabbits. The scars of the treated rabbits were stronger and larger, being composed of dense connective tissue with fibers arranged in a more normal, parallel fashion.

A more recent study, completed by Chi-Sing et al⁴⁴ in 1991, used 120 male Hartley guinea pigs. A scald burn was induced on their backs, covering 8 to 9 percent of their TBSA. These wounds were then grafted. A constant direct current was applied to the wounds for five days. The results showed that epithelial growth was distinctly stimulated by direct current and microcirculation was enhanced.

Carley and Wainapel⁴⁵ studied patients in 30 hospital settings. Wound healing protocols used LIDC. The patient's wound was stimulated with the cathode for three days followed by the anode until the wound healing

plateaued. At that time, the cathode was used again for three days. Results were the LIDC group healed 1.5 to 2.5 times faster. Another effect was the overall subjective complaints of pain and discomfort were decreased when compared to the control group. The authors' logic for using the cathode and then the anode was that the cathode had bactericidal effects and the anode tissue growth effects. This concept is consistent regardless of what type of stimulation is used for wound healing.^{39,46}

High Voltage, Monophasic, Pulsed Current (HVPC)

HVPC is electrical stimulation using a twin peaked pulse duration, measured in microseconds (μsec) and with a peak current of up to 2500 milliamps (mA). It is used to enhance wound healing.

McDonnell and Menton⁴⁷ made an incision wound on 36 rabbits. Half of the rabbits were treated with HVPC for four hours per day. Epithelialization started sooner and more collagen was present in the HVPC group in relation to the control group.

Cruz, Bayron, and Suarez⁴⁸ burned 20 domestic pigs and applied HVPC to ten of the pigs for ten minutes daily. Results concurred with the other studies by showing faster healing rates in the treatment group.

Kloth and Feedar⁴⁰ studied 16 patients with various wound types.⁴⁰ Their study agreed with the animal studies: electricity enhances the rate and extent of wound healing.

Only one study was found to refute all of the studies mentioned. Gogia et al⁴⁹ used HVPC on 12 patients. There was no significant increase in the healing rate in the treated group as compared to the control group. The lack of significance was felt to be due to the variability of the parameters of stimulation and the small group size.

Most studies cited above showed accelerated healing and increased strength of the new tissue, but this has been shown to be significant only in the early stages. After closure, the tensile strength of the scar of electrically stimulated wounds may not be any different than that of control wounds.⁴⁷

Transcutaneous Electrical Stimulation (TENS)

Electrical stimulation has been researched for its ability to decrease the pain perception of patients. Specifically, Transcutaneous Electrical Stimulation (TENS) has been investigated for its ability to decrease pain in two ways, using the gate control theory and the endogenous opiate theory.⁵⁰

TENS units can be adjusted for various sensations and purposes. Low TENS, also called acupuncture-like TENS, has a short frequency of one to four pulses per second (pps) and a large duration of 150 to 250 μ sec. The intensity is set to produce a visible muscle contraction within the patient's tolerance. The treatment time is 20 to 30 minutes. Low TENS is thought to produce a prolonged analgesic effect by stimulating the release of endorphins in the central nervous system.⁵¹

A pilot study by Lewis et al⁵² experimented with the use of acupuncture-like TENS. Auricular low TENS was performed on burn patients. Stimulation of the external ear in various sites is based on the premise that there is an orderly somatotopic representation of the body surface and visceral structures which resembles an inverted fetus. Subjective pain levels in comparison to controls were significantly reduced.

High TENS, also called conventional TENS, has a higher frequency ranging from 50 to 100 pps with a shorter duration of 20 to 60 μ sec. This is a sensory only modality, producing a paresthesia-like sensation without a visible muscle contraction. It is said to have a shorter lasting effect than low TENS and is based on the gate control theory of pain control.⁵⁰

Contraindications of Electrical Stimulation

There are two contraindications for the use of electrical stimulation. If the patient has a cardiac pacemaker or if the stimulation was to be over a pregnant uterus, electrical stimulation for wound healing or pain control should not be done.⁵⁰

In conclusion, several studies show electrical stimulation can be of benefit. A major drawback is that there are no set protocols for its use. The chances of success by lay-therapists are probably reduced.

A final point is this: if the long-term results are not significantly better than conventional treatment, it may be unethical to charge the patient for

treatment which is not beneficial in the long run. This point should also be thought of with the use of lasers that will be discussed in the next chapter.

CHAPTER VI

LASERS

This chapter will focus on the use of lasers for wound healing and pain management. It will begin with a review of laser history and characteristics and progress to findings relative to wound healing and pain management. Finally, precautions and contraindications for lasers will be addressed.

History and Characteristics

LASER is an acronym for Light Amplification by Stimulated Emission of Radiation.^{53,54,55,56} In 1892, smallpox, as was also noted with electrical stimulation, was one of the first diseases treated with therapeutic light.^{54,57} Neils Ryberg Finson⁵⁴ supported the use of red light on the smallpox lesions to decrease the inflammation, foster healing, and decrease the scarring. In 1960, Dr. Theodore Maiman^{53,55} brought the quantum physics of Einstein to reality by exciting a ruby rod crystal with intense pulses of light from a xenon flash lamp. While the ruby red laser is limited to the visible red light spectrum, research and development have led to lasers which produce radiation in the infrared, visible, ultraviolet, and X-ray spectrums.⁵³ In the 1990s lasers have become a standard part of medical treatment.⁵⁵

High power lasers used for surgical procedures have output of 10 to more than 100 Watts (W). Low intensity lasers have an output less than 50 milliwatts (mW).⁵³ Low level lasers have been called cold laser, soft laser, laser biostimulation, and low reactive-level laser therapy.⁵⁷ Low level stimulation is best suited for physical therapy purposes because of the "Arndt Shultz Law."⁵³ This law claims that strong stimuli retard physiologic activity and weak stimuli accelerate physiologic activity.⁵³

The type of laser is determined by the wavelength of the light used, which is dependent upon the medium used to produce that light. The lasers used for physical therapy purposes are in the visible red and near infrared spectrums.⁵⁸ The most common mediums for physical therapy laser are: argon, helium-neon (HeNe), ruby, gallium-arsenide (GaAs), and gallium-aluminum-arsenide (GaAlAs).⁵⁷

Before proceeding to findings of research, it is important to mention that the low-level laser therapy used with physical therapy has not been approved by the FDA in the United States. In Europe it is used as a treatment, but in the U.S. it is only used for research purposes.

Wound Healing Studies

Laser light reportedly increases the rate of tissue regeneration. Specifically, it has been noted to activate phagocytosis, increase the granulation rate, and cause faster epithelization of wounds.^{51,59} All of these effects are very controversial due to variable results in research studies.^{53,57,60,61,62,63,64} Wound-

healing experiments have shown acceleration of healing, but these results are often in early phases and have conflicting results.⁶³

Separate case reports regarding laser treatment for a diabetic ulcer and lacerations revealed that HeNe laser enhanced the healing of both wounds.⁶⁰ Kovinskii⁵⁹ reported that the use of HeNe lasers with human burn wounds was beneficial.

Gogia et al⁶⁴ studied twelve patients with chronic lower leg/foot ulcers. Thesis results showed no significant acceleration of healing after the use of HeNe laser; low level stimulation was used.

Basford et al⁶² studied six pigs that had full thickness wounds created on each side of their spines. The study did not show significantly altered healing in comparison to its controls. Again, low level of stimulation was used; higher treatment energies may have produced significant increases in wound healing.

Pain Management Studies

The research regarding analgesia with laser treatment is proven, but how this effect occurs is controversial. Despite the skepticism, research has shown 70 to 80% of those treated with this modality have partial or total relief from pain.⁵³ Pain relief has been found to result from an "overall systemic effect."⁶³ Inflammation decreases in the area of the wound which in turn lessens pain by removing the pressure in the free nerve endings and pain receptors. Goldman et al⁵⁶, Asada et al⁵⁶, and Bliddal et al⁵⁶ studied people with rheumatoid arthritis

and found positive results. The only drawback from these studies is that each one used a different set of parameters.

Precautions and Contraindications

As with all other modalities, there are certain safeguards of which one must be aware. The main precaution is that no one should stare directly into the laser beam.^{54,58} The therapist and patient should wear protective goggles during the treatment session.

There are some very important contraindications. These include: pregnancy, fontanelles of growing children, photosensitive patients, the eye, neoplastic tissue, cancer, hemorrhaging regions, obtunded reflexes, the gonads, infected tissue, sympathetic ganglia, vagus nerves, and the cardiac region of the chest in patients with heart disease.^{54,58}

In conclusion, this chapter has explained the history and characteristics of lasers, reviewed only a few of the studies of laser usage for wound healing and pain management, and explained precautions and contraindications. This modality could be of benefit in the United States, but its use is severely limited by the lack of approval of the FDA. U.S. studies have had variable results using one joule or less. European treatments using higher energy outputs of one to four joules have shown good results.

CHAPTER VII

CONCLUSION

This paper has briefly touched on the areas of wound healing and pain management in the patient with burns using continuous passive motion, electrical stimulation, and lasers. To conclude, there are two points the author wishes to express about the results of this literature review.

The first point is that there are limited resources for the topic, especially in the English language. Thus, more research must be put into these modalities if they are to be used more frequently in the United States.

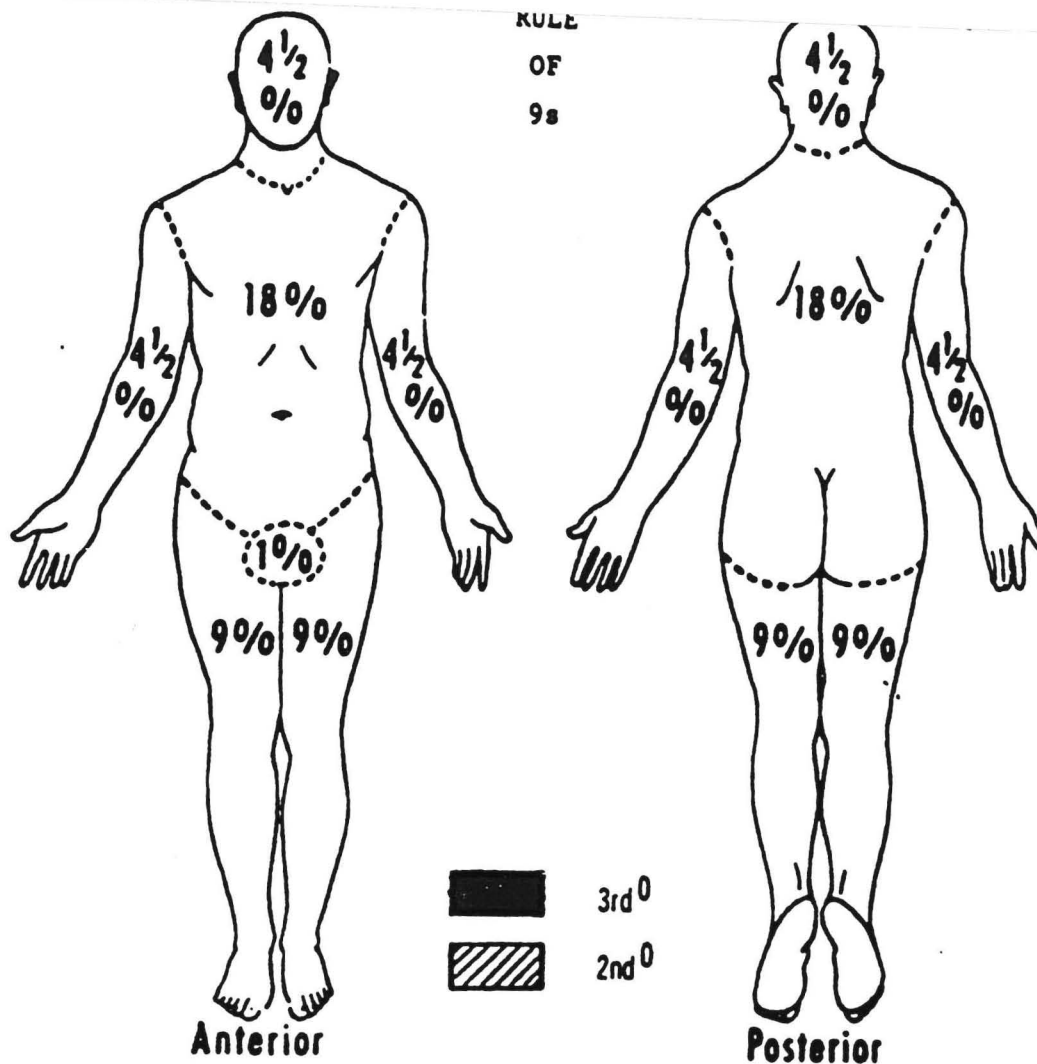
Secondly, as the author was reading each study, it was hard to carry information from one article to the next. One reason for this problem was a lack of consistency in the naming of parameters, such as laser pulses per second versus total treatment joules, and variance of dosages. These discrepancies help explain why these modalities have not been proven to have a positive effect.

With Health Care Reform looming over the shoulders of physical therapy, there will be a time when treatments will only be reimbursed that are backed by scientific proof. In the future, researchers need to be more specific in their parameters and dosages. Once a modality is proven effective, research should

be more directed toward the most efficient protocol to obtain that desired outcome.

APPENDIX A

** From: Richard RL, Staley MJ. Burn Care and Rehabilitation: Principles and Practice. F.A. Davis, Philadelphia, PA, Fall, 1993.



LUND AND BROWDER CHART

AREA	inf	1-4	5-9	10-14	15	adult	part.	full	total	Donor areas
Head	19	17	13	11	9	7				
Neck	2	2	2	2	2	2				
Ant Trunk	13	13	13	13	13	13				
Post Trunk	13	13	13	13	13	13				
R. Buttock	2†	2†	2†	2†	2†	2†				
L. Buttock	2†	2†	2†	2†	2†	2†				
Genitalia	1	1	1	1	1	1				
R.U. Arm	4	4	4	4	4	4				
L.U. Arm	4	4	4	4	4	4				
R.L. Arm	3	3	3	3	3	3				
L.L. Arm	3	3	3	3	3	3				
R. Hand	2†	2†	2†	2†	2†	2†				
L. Hand	2†	2†	2†	2†	2†	2†				
R. Thigh	5†	6†	8	8†	9	9†				
L. Thigh	5†	6†	8	8†	9	9†				
R. Leg	5	5	5†	6	6†	7				
L. Leg	5	5	5†	6	6†	7				
R. Foot	3†	3†	3†	3†	3†	3†				
L. Foot	3†	3†	3†	3†	3†	3†				
TOTAL										

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